Results: Compared with essential hypertensive patients, those with hyperaldosteronism were younger (56.9 [11.7] years vs 60 [14.4] years; P<.001), had higher blood pressure prior to the etiological diagnosis (156 [23.2] mmHg vs 136 [20.6] mmHg), more frequently had a family history of early cardiovascular disease (26.7% vs 2.3%; P<.001), and had a higher prevalence of concentric left ventricular hypertrophy (72% vs 25.4%) and higher cardiovascular risk. Specific treatment resulted in optimal control of systolic and diastolic blood pressures (from 150.7 [23.0] mmHg and 86.15 [14.07] mmHg to 12.69 [15.3] mmHg and 76.34 [9.7] mmHg, respectively). We suspected the presence of hyperaldosteronism because of resistant hypertension (38.6%), hypokalemia (39.7%), and hypertensive crises (12.7%). Only 4.3% of these patients had been referred from primary care with a suspected diagnosis of hyperaldosteronism.

Conclusions: Hyperaldosteronism should be suspected in cases of resistant hypertension, hypokalemia and hypertensive crises. The diagnosis of hyperaldosteronism allows better blood pressure control. The most prevalent target organ damage is left ventricular hypertrophy.

0194

Prospective analysis of plasma cholesterol and triglycerides in patients (pts) with chronic phase (CP)-chronic myeloid leukemia (CML) during treatment with the 2nd generation tyrosine kinase inhibitor

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Despite a well-recognized clinical benefit of the 2nd generation tyrosine kinase inhibitor nilotinib in patients with imatinib-resistant/-intolerant or newly diagnosed chronic myeloid leukemia, recent evidence suggests that nilotinib has a propensity to increase the risk of occlusive arterial events, especially in patients with pre-existing cardiovascular risk factors. Given the key role of lipids in cardiovascular diseases, we studied the plasma lipid profile and global cardiovascular risk prior to and during nilotinib therapy in a series of 27 patients in the setting of an observationalsingle-center study. Data from a minimum 1-year follow-up showed that nilotinib significantly increased total, low and highdensity lipoprotein cholesterol within 3 months. Consequently, the proportion of patients with non-optimal low density lipoprotein cholesterol increased from 48.1% to 88.9% by 12 months, leading to cholesterol-lowering drug intervention in 22.2% of patients. The proportion of patients with low levels of high density lipoprotein cholesterol decreased from 40.7% to 7.4% by 12 months. In contrast, a significant decrease in triglycerides was observed. Global cardiovascular risk worsened in 11.1% of patients due to diabetes or occlusive arterial events. Whether hypercholesterolemia was the main driver of occlusive arterial events was uncertain: a longer follow-up is necessary to ask whether nilotinib-induced hypercholesterolemia increaseslong-term risk of atherosclerotic diseases. Nevertheless, given keyatherogenicproperties of low density lipoprotein cholesterol, we conclude that when prescribing nilotinib, commitment to detect lipid disordersat baseline and during follow-up is mandatory given their frequency, requirement for lifestyle or drug intervention and potential for long-term cardiovascular complications.

0195

Identification of patients (pts) with chronic myeloid leukemia (CML) at high risk of artery occlusive events (AOE) during treatment with the 2nd generation tyrosine kinase inhibitor (TKI) nilotinib

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Background: Nilotinib is approved for use in pts with CML after failure of imatinib and in newly diagnosed CP-CML. However, several studies report a nilotinib-associated risk of AOE (arterial occlusive event), especially in pts with preexisting risk factors for CVD. In this study, we aimed at determining whether CVD risk estimation using the 2012 ESC classification could be useful to identify patients at high risk of AOE during nilotinib therapy.

Methods: Pts (n=75) treated with nilotinib upfront or after failure of prior TKI at our institution were included provided that baseline CVD status could be retrospectively collected. Patients were categorized into 2 groups according to ESC 2012 classification: low/moderate (L/M) and high/very high (H/VH) CVD risk.

Results: At nilotinib initiation, median age was 51 years (19-76), 41 pts (54.7%) were males. At baseline, medical history revealed H/VH risk category in 15 pts (20%) including established CVD in 6 pts (8%) (all diagnosed before CML), DM (diabete melitus) in 10 pts (13.3%), severe AH (arterial hypertension) in 1 pt (1.3%), familial dyslipidemia in 1 pt (1.3%) and a SCORE ≥5% in 2 pts (2.6%).

AOE occurred in 12 pts with myocardial infarction (MI) or coronary heart disease (CHD) (n=3), cerebrovascular events (CeVD) (n=3) and peripheral artery disease (PAD) (n=6). Cumulative incidence of AOE by 48 months was 72.22% (95% CI: 47.46-100) in the H/VH group and only 12.13% (95% CI: 4.32-34.08) in the L/M group. Log Rank comparison of Kaplan Meier analysis of 48-month survival without AOE showed a significant difference between the 2 groups (27.78% (95% CI: 0-58.9) versus 84.38% (95% CI: 67.04-100) p=0.0001). Sensitivity of the ESC classification in nilotinib-treated patients was 67% and specificity 89%.

Conclusions: In our retrospective study, CVD risk estimation according to the 2012 ESC classification reveals that pts who belong to the H/VH risk group at baseline are at very high risk of AOE during nilotinib therapy. In this context, CVD risk should be reassessed throughout therapy and risk factors should be tightly controlled according to current guidelines.

0270

Arterial involvement in Behcet's disease: a series of 20 cases

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Background: While venous manifestations are frequent and have been reported in many publications, data regarding arterial lesions in patients with Behçet's disease (BD) are rare and often isolated.

Aim: We report our experience with a rare and interesting subset of BD patients with arterial involvement

Methods: From 1998 to 2013, 20 cases of arterial involvement were found amongst BD patients in the department of internal medicine of Hedi Chaker Hospital. All these patients fulfilled International Study Group criteria for BD.

Results: The mean age when arterial involvement manifested was diagnosed was 32.9±9.9 years, and 18 of the patients were male. Combination of venous and arterial manifestations occurred in 10 patients The artery most often affected is the pulmonary arteries (11 cases) followed by the aorta, the femoral arteries (5 cases) and the coronary arteries (4 cases). The arterial disease was the presenting symptom of Behcet's disease in 13 cases. Arterial lesions included arterial thromboses (45%), aneurysms (30%), stenosis (20%) and Coronaritis (5%). 15 patients underwent corticosteroids and 4 patients underwent immunosuppressive therapy. Three patients were successfully operated for aneurysms. After a median follow-up of 66.9± 54.4 months, 3 patients had died, in 2 cases directly related to cardiac involvement.

Conclusion: Arterial findings are not uncommon in BD. Assessment of the cardiovascular system is advised in patients with BD even if there is no clinically apparent cardiovascular disease.

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